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DISEASES OF THE CHEST

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Editorial Comment

RED CROSS ROLL CALL

THE Roll Call Season is approaching, and it is well for us to remember that for the past 55 years the flag of the Red Cross has flown upon every scene of major disaster in the United States. The Red Cross has carried assistance in the form of food, clothing, shelter, medical aid, rehabilitation of homes and families to more than 1200 scenes of disaster, large and small.

American people through this Society and the people who support it, and actively work for it through the years, have saved thousands of lives, prevented many major epidemics of disease, and given a helping hand to those left hopeless and homeless by the forces of nature.

To the public mind the Red Cross Nurse has come to be accepted as a symbol of mercy. There never has been a shortage of physicians in any disaster or emergency. Medical men always offer their services to the Red Cross in time of need.

The neighborly spirit which rules over America has, since the colonial days, constituted one of the bulwarks on which our country has grown to greatness. It permeates life in rural sections, villages, towns, and cities alike. Neighborliness is a trait inherently American, and at no time does this become more evident than during periods of great stress and calamity. It is truly American be a Volunteer in the American Red Cross. *Join now.*

C. M. H.

TUBERCULOSIS AND ORGANIZED MEDICINE—The Philadelphia Plan

ON October 6th, 1937, in Philadelphia, Resolutions were presented and passed by the Governing Body of the Pennsylvania State Medical Society to appoint a Tuberculosis Committee to cooperate with the Tuberculosis Committees of the County Medical Societies of the State.

This is the opening gun in a campaign to secure a Tuberculosis Committee in every unit of organized medicine—Municipal, County, and State Medical Societies and, we hope, in the American Medical Association.

The Crux of the Philadelphia Plan is *Balanced* Activity. There are *Three* Great Phases in Tuberculosis Work—Case Finding, Case Treatment, and Rehabilitation. They should be *equally* stressed. What is the use of finding a case if the case cannot secure proper treatment? What good is proper treatment if the patient must relapse through unsuitable labor when returning to industry?

The need of Organized Medicine in the Tuberculosis Fight has always existed, but has become imperative since the ad-

vent of active treatment of the tuberculous patient with Collapse Therapy.

The work of studying and treating Tuberculous patients in Sanatoria is so extensive and time-consuming, that it is necessary to have one full time physician to fifty patients. In Pennsylvania State Sanatoriums there is one doctor to treat about 250 patients. It is necessary for a pneumothorax patient to be studied and treated by an individual physician, one experienced in pulmonary disease in general and pneumothorax treatment in particular. In some of our Sanatoriums and Hospitals, due to insufficient medical personnel and other causes, the patients are bandied about from one doctor to another, seldom receiving consideration from the same doctor twice in succession. Still worse, in some Hospitals the living tuberculous patients are used as cadavers to furnish experience to internes who have no desire or intention of becoming chest specialists, but are just taking a rotating service to fit them for general practice. All such "training" can do, besides injuring the patient, is to give the interne that "little knowledge" which is a "dangerous thing."

ORGANIZED MEDICINE is *needed* to *bring influence* to *bear upon Public Health Authorities* to see *that the Case has a chance of being properly treated after* it is *found.*

The Tuberculosis Committee of the County Medical Society is the unit of this organization. In Philadelphia, the set-up is as follows:

Tuberculosis Committee:

Frank Walton Burge, M.D., Chairman;

Sub-committees for Case Finding:

Esmond R. Long, M.D., Chairman; Joseph McEldowney, M.D.; Robert G. Torrey, M.D.

Sanatorium Investigation:

A. J. Cohen, M.D., Chairman; Esmond R. Long, M.D.; Henry M. Minton, M.D.

Rehabilitation:

Robert G. Torrey, M.D., Chairman; Burgess Lee Gordon, M.D.; Charles A. Heiken, M.D.

Racial Protection:

Henry M. Minton, M.D., Chairman; Moses Behrend, M.D.; William G. Turnbull, M.D.

Bed Rotation:

William G. Turnbull, M.D., Chairman; Moses Behrend, M.D.; Robert G. Torrey, M.D.

Cooperative:

Moses Behrend, M.D., Chairman; A. J. Cohen, M.D.; Joseph McEldowney, M.D.

T. B. Nursing:

Joseph McEldowney, M.D., Chairman; A. J. Cohen, M.D.; Burgess Lee Gordon, M.D.

The work of most of these subcommittees requires no explanation.

The Racial Protection Subcommittee will work to secure a change in the plan now in use for admitting patients to sanatoria, so that the death rate from tuberculosis in any race will be included along with the number of that race in allottment of beds.

The Bed Rotation Subcommittee will endeavor to secure exchange of patients between Mountain Sanatoriums and City Tuberculosis Hospitals so that the curable cases are given climatic advantage and the old and incurable chronic cases are near home where they can enjoy visits from their loved ones.

The Cooperative Subcommittee will work with the Cooperative Committees of all other Counties and with the State Committee to secure a Tuberculosis Committee in every County Medical Society in the State.

The T. B. Nursing Subcommittee will work to secure reestablishment of the nurses training schools in tuberculosis sanatoriums and to assist with arrangement with hospitals for completion of their training. The Sanatorium Trained Nurse has been the backbone of Tuberculosis Nursing in Sanatoriums and the loss of new graduates since the abolition of such training schools is being badly felt; besides, such training is the finest Rehabilitation for many of our highest type female tuberculous patients.

The Philadelphia Plan is just forming. We beg for constructive ideas and criticisms from the whole World. F. W. B.

BLOOD EXPECTORATION It would seem by now that every one concerned with the care and handling of the sick would fully realize and appreciate the significance of the expectoration or coughing up of blood. In too many cases however this occurrence is being attributed to a hemorrhage from the throat, the benign rupture of a capillary in the bronchial tube, or to some other erroneous explanation. This false or mistaken explanation may be comforting to the patient for the time being; but usually invites tragedy later. It permits the morbid process in the lungs to advance a pace, and the optimum time for a cure to slip by. It is well and wise to remember that most instances of coughing up blood, however small in amount, are most often due to disease in the lungs. This disease, in the vast majority of cases, is tuberculosis.

The expectoration of blood is also seen with some frequency in bronchiectasis, lung abcess, carcinoma and in some patients with valvular heart disease. As some figures have it: 90 per cent of all obscure blood spitting comes from the lungs; and when from the lungs, 95 per cent is due to pulmonary tuberculosis. These figures are impressive and should carry a vital warning. To be safe and also wise, consider all cases of obscure blood spitting as due to tuberculosis until proved otherwise.

The term obscure is used to eliminate cases of trauma, recent extraction of teeth, operations on nose and throat, etc. For a throat to bleed, except in instances of trauma or following operative proceedures, is rare. *Remember this!* Realize also that hemoptysis, the coughing or spitting up of blood, blood streaked or blood stained sputum, is a symptom of extreme significance and demands a satisfactory determination of its origin. This origin in most instances is in the lungs, and the cause most often pulmonary tuberculosis. C. H. H.

The October Indiana Public Health Bulletin reports the following deaths for August: Tuberculosis 107 vs. Syphilis 17.

SPREADING HEALTH EDUCATION In a recent survey conducted by the Committee on Circulation for Tuberculosis Publications of the Associated Editors of Tuberculosis Publications, it is interesting to note that 34 tuberculosis periodicals published in the United States and Canada report a combined monthly circulation of 118,040 copies and 1,416,480 copies annually. These figures do not represent all of the tuberculosis publications issued in North America, for many of the periodicals did not report their circulations.

Of the 34 publications reporting, "The Chaser" issued by the State Tuberculosis Sanatorium of Texas has the largest individual circulation with 50,000 copies monthly. "The Town Crier" of the Muirdale Sanatorium, Wauwatosa, Wisconsin, has the smallest number of copies, 185 issued bi-monthly. The reporting periodicals included 28 issued monthly, 4 bimonthly, 1 weekly, and 1 quarterly. Of these 29 are published in the United States and 5 in Canada.

Featured in most of the publications are scientific articles on all phases of the prevention, treatment, and history of tuberculosis; inspirational articles by patients and ex-patients; cartoons of interest to patients and others, and biographies of persons who have become famous despite the handicap of tuberculosis.

This vast amount of literature circulated in most cases by non-profit groups is an important link in the educational program against the ravages of tuberculosis. Not only do they serve as a medium of occupational therapy for patients, but these publications reach into the homes of several hundred thousand families in this country and in Canada. Their beneficial influence in combatting tuberculosis and in spreading health education should not be underestimated.

Committee conducting Survey: Ethel Hale Cox, State Sanatorium, Arkansas, Chairman; Mary J. Fitzmaurice, Laurel Hill, Secaucus, New Jersey; Mary Jo Patty, State Sanatorium, Rockville, Indiana; and Jean Paul Talbot, Fort Sanatorium, Saskatchewan, Canada. C. M. H.

Tuberculosis: A Retrospect and Forward *

BY

WM. EGBERT ROBERTSON, M.D.**

Philadelphia, Penna.

THE earliest references to medicine are largely legendary. Biblical descriptions are not very precise. Many learned men have attempted to trace the history of syphilis in the Bible, but without success. It seems possible at times that it is clouded by the general comments on leprosy. Neuberger has said quite appropriately that the chief glory of Biblical Medicine exists in the presentation of social hygiene as a science. The Mosaic Laws as set forth in Exodus and Leviticus may be accepted as the origin of medical jurisprudence, and, I might add with propriety, Jewish prudence. The old Hebrew ban put upon sexual promiscuity and perversions, and restrictions placed upon sexual congress, governed by the menstrual period, all served to develop self control and continence. At the same time it apotheosized virtue, throwing a mantle of chastity and veneration about womanhood, which has become a dominant trait among all civilized people, reaching down to the present day. The Laws of Moses as portrayed in the Pentateuch, the Torah of the Hebrews, constitute the written word. The story teller and soothsayer are equally responsible for much that has descended upon us from the dim past. The Talmud for instance. This began essentially as word of mouth traditions after the Babylonian Captivity, about 536 B. C. Gradually accumulating, they became the law as set forth in the Mishna, the explanations and commentaries being that part known as the Gemara. This work as the written word of the Law, came into being about the second century A. D., and both the Babylonian and Palestinian Talmud include references to medicine as understood by the Jews. Apparently the earliest reference to hemophilia occurs in the Babylonian Talmud. Dispensation was granted, when, in connection with circumcision, two successive children in a family died of a fatal hemorrhage. This was regarded as a familial, hemorrhagic disease. The next reference occurs in the work by Albucasis, nine centuries after the Talmudic reference, and not again until 1802, a lapse of eight centuries, when John C. Otto of Philadelphia published his article in the Medical Repository.

Prior to the advent of Andreas Vesalius, no trustworthy descriptive work in anatomy existed. The general horror of touching and desecrating the dead, put a ban upon human dissections, hence such knowledge as existed in the pre-Vesalian period, grew up about the social, hygienic laws of the Jews and the inspection of meat to be used for food, as to whether it was Kosher or trepha, or tref as it is equally well known, clean or unclean. The Talmud contains a detailed description of the esophagus, trachea and larynx and includes comments on hyperemia, caseation and tumors of the lungs, yet it is said that there is no word in the Hebrew tongue for cough or phthisis.

The lungs from early times were recognized as air containers, but they were supposed at the same time to deal with spirits which had been concocted in the heart. Because of the strict interpretation of Kosher, the Talmud ordered the refusal of the carcas of any animal for food, when the lung failed to retain air upon inflation. This led naturally to an appreciation of the pleura in relation to possible disease, and to the bronchi. As a matter of fact, up to the time of the publication in 1661 of the studies of Marcello Malpighii "On the Lungs," little was known concerning the structure of the lung. It was described as fleshy and porous, a structure in which the smaller divisions of blood vessels and the ramifications of the bronchi were lost. The pulmonary circulation was first described

*Delivered before the American College of Chest Physicians at Atlantic City, June 7th, 1937.

**President-elect, Philadelphia Medical Society.

by Michael Servetus in a religious work entitled "Restitutio Christianismi" in 1553, the same year in which he was burned at the stake at the behest of John Calvin. Servetus stoutly denied the existence of pores in the septum of the heart for the passage of blood and spirits, but he accepted the doctrine of a vital spirit imparted to the blood in its passage through the lungs. In a sense this was true, but it required the labors of many men even up to comparatively recent times, before the composition of air became known. This banished the older doctrines of spirits and fuliginous vapors.

It was the introduction of the compound lens microscope in 1625, that aided Malpighii to discover the capillaries in the lung which he described in 1661, thus with the work of Servetus, completing the discovery of the circulation of the blood published by William Harvey in 1628. The function of the lungs as we understand it today, was a matter of gradual development, as previously stated, dependent upon the discovery that the air is a composite substance. It is a history in itself, too long and detailed to receive further notice now.

The Hippocratic facies and Hippocratic fingers attest to the recognition of pulmonary tuberculosis as early as the fourth century B. C. As a clinical entity it was known to the Greeks. Galen believed it to be contagious. Suffice it to say that the knowledge of tuberculosis as a pathological entity is relatively recent. It was Franciscus de le Boe, known as Sylvius, who first pointed out in the seventeenth century, the relation of the tuberculous nodule to phthisis. To Matthew Baillie of the eighteenth century, Bayle and his pupil Laennec, and especially to Laennec, both of the nineteenth century, we owe our present knowledge of tuberculosis, and to Laennec, the introduction of the stethoscope in 1819, and thus the foundation of modern clinical medicine. Villmin's experimental work published in 1865 was the first proof of its inoculability. He said, "Tuberculosis is the effect of a specific causal agent, a virus." It remained for Koch to discover the tubercle bacillus in 1882.

Air and fluid in the pleural cavity must have been recognized by Hippocrates if we accept the so-called Hippocratic succussion splash. Celsus in the first century of the Christian era, recommended the introduction of a hot iron for the evacuation of fluid. Purulent and other fluids in the pleural cavities have been recognized entities for centuries, but they were ascribed to punctured wounds or to fracture of the ribs. The first attempt, experimentally to produce artificial pneumothorax in rabbits and dogs is ascribed to William Hewson, whose work was published in 1767. At the same time he recommended paracentesis as a therapeutic measure for air in the chest. This led to a controversy with his former teacher, Alexander Monro, the younger. This controversy is set forth in detail in the life and work of William Hewson, published by the Sydenham Society in 1846 and 1847. Munro also laid claim to the work of Hewson on the lymphatics in animals. The lymphatics were originally discovered in man in 1653 by Olaus Rubeck, a Hollander, after whom the Rudbeckia is named. The word pneumothorax was coined by Itard, a French student, in a Thesis submitted for the degree in medicine in 1803. Hewson refers to air in the chest, and such was the designation prior to Itard's Thesis. The practice of artificial pneumothorax is said by Garrison in his History of Medicine, to have been suggested by James Carson in 1842. The only corroboration of this I found in German literature. Carson was a Scotch physician, who practised in Liverpool, England. However, Carlo Forlanini is regarded as the one who first performed artificial pneumothorax. For some time it has been observed that the presence of air or fluid in the thorax exercised a more or less restraining influence upon the course of pulmonary tuberculosis. Probably this led Forlanini in 1882 to suggest artificial pneumothorax as a therapeutic measure, though he did not practice it till 1888, and his paper was not published until 1890.

According to Heynsius, Donders was

the first to measure intrapleural pressure. Seemingly unaware of the work of Forlanini, John B. Murphy and his assistant van Lowki introduced the technique to the United States in 1898 and von Brauer to Germany in 1906. Positive pressure and more or less total collapse were practiced at first, but partial or selective pressure, popularized especially by Ascoli in 1912, has greatly increased the range of usefulness, even to the performance of bilateral compression. More formidable but of great value in special cases, is the operation of thoracoplasty, proposed by Brauer but first performed by Friedrichs on December 11, 1907. Brauer also devised and first performed cardiolysis. Similar decortication of the lung, Delorme's (1894) or Fowler's operation (1901), Jacobaeus pneumolysis, local thoracoplasty of Quincke and Wilms thoracoplasty are more or less felated methods, having for their object, fundamentally, the eventual collapse and complete inactivity of involved lung tissue.

Allied too, is the Tuffier operation or lipotamponade, applicable only to cases of tuberculosis. It is probable that Hilton's famous book on "Rest and Pain" has played a part in stimulating many of the measures now more or less routinely applicable to the different stages and varieties of pulmonary tuberculous infection. This is equally true of phrenicotomy and its variants, first suggested by Stuertz and performed by him in 1911, and independently by Sauerbruch. Whether lobectomy or excision of an entire lung will still be regarded as an operation of choice, when one has so many therapeutic gradients of a less destructive character to choose from, must remain for future determination. The practical deduction is that the management of tuberculosis has become a group responsibility of the dietition, the laboratory, the x-ray department, the trained physician and the trained surgeon. Until specific therapeutic measures have been discovered and the general physician has learned to recognize the incipient case, the chest clinic must be regarded as the clearing house and the sanitarium as the repository. It seems evident too that the sanitarium cannot become a complete and satisfactory entity without a trained surgical department. Of this I was never more strongly convinced than when I had the privilege of introducing two of the medical and surgical clinicians of the department of tuberculosis of the Philadelphia General Hospital, at our recent Postgraduate Assembly at Philadelphia. Dr. Heiken discussed the applicability of pneumothorax, and Dr. Behrend, chief of the surgical department for the tuberculous, presented a number of recovered cases, some of them self-supporting, restored by thoracoplasties, one of them a patient who had been successfully operated upon by Dr. Astley. This clinical demonstration furnished proof of the value of team work in the study and management of the tuberculous individual.

Before concluding, may I refer briefly to the heart in the tuberculous patient, omitting reference to tuberculous pericarditis. Most tuberculous individuals have a small, hypoplastic, vertically placed heart and a diaphragm occupying a low position. This is especially true when they are also visceroptotic. Examination of the abdomen, and even fluoroscopy after a barium meal, should be a routine in every case suspected as one of tuberculosis. An abdominal support for those ambulatory, and elevation of the foot of the bed for those who are confined to bed, will eliminate many of the gastrointestinal, digestive upsets and enhance nutrition, at the same time lessening the work of the heart. In the more advanced case of pulmonary tuberculosis, it is the burden which is thrown upon the right ventricle, the result of increased pressure in the pulmonary circulation due to pleural adhesions, destructive ulcerative lesions of the parenchyma and fibrosis. The management of such a heart depends upon the team work previously referred to, the proper handling of the tuberculous process and not the exhibition of digitalis, especially when the patient is febrile. It was Rokitansky who called attention to the great infrequency of tuberculosis in an individual with mi-

tral stenosis whatever the explanation may be. Valvular lesions of any character are the exception in the tuberculous and then a coincidental occurrence, rather than cause and effect. Most cases of tuberculosis are prone to present dominant right heart phenomena, or with the advance of the disease, progressive failure of the cardiac musculature in toto, and edema. The edema is of ill omen though rarely wide spread. Actual tuberculosis of the heart muscle or valves, is rare. Even in miliary tuberculosis, the grandines, as the miliary tubercles were called some years ago, are rarely found in the heart muscle, but do not cause symptoms when they are present. It is conceivable, of course, that symptoms would arise if any part of the conducting system or nodes were involved. Rarely a caseous, solitary tubercle has been reported, causing aneurysmal dilation and rupture. There is no doubt that pulmonary tuberculosis makes serious inroads upon the cardiovascular system, but the management of such a condition is essentially the management of the underlying tuberculosis.

Congenital Cystic Disease of the Lungs

(A Review of Literature and Case Report)

BY
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Sanatorium, Miss.

As long ago as 1638 Nicholus Fontaine examined the body of a three months old baby, dying of respiratory failure, and reported cysts or air blebs scattered throughout both lungs, but Koontz[1] in 1925 probably made the first report, in this country, of cystic disease of the lungs. He made an exhaustive study of the literature and was able to find 108 cases —all of which had been diagnosed at autopsy findings and reported by European physicians. Most of the cases cited were by German and English doctors. Meyer[2] in 1859, is credited with the earliest clinical case, which was a solitary cyst. Croswell and King[3] in 1933, gave an excellent review of a three year old child having an enormous cyst which filled the entire left chest and herniated through the anterior superior mediastinum to the right upper lung. In spite of the gravity of the condition the child survived. Since this last article many cases have appeared in the medical literature. In fact, I have found a total of sixty-seven American cases, including the following one.

A white man, farmer, age 22, was admitted to Catawba Sanatorium May 12, 1936 with a complaint of cough and expectoration during his entire life. The family history is essentially negative. Father died of influenza in 1918; one brother and two sisters, alive and well. There is no history of tuberculosis nor of other contagious diseases in the family.

The past history is interesting. There were the usual childhood diseases — whooping cough at 5, chicken-pox at 6, and measles at 8 with good recoveries. However, he gives a history of pneumonia at 3, 15, 16, and 17. During one attack he became extremely ill and was not expected to live. At no time did the left chest escape the infection, and at the ages of 3 and 17 he had bilateral pneumonia. Aspiration was attempted during the last attack but no fluid obtained. At the ages of 18 and 20 years he developed pleurisy, left chest, which lasted about two weeks each time. He has had cough and expectoration most of his life and at times the sputum became offensive. He also had frequent attacks of shortness of breath and a sensation of tightness in his chest. His mother noticed a wheezing of the patient at birth. Several physicians have examined him during his life and all have made a diagnoses of "bronchial trouble" —only one physician thought the malady

tuberculosis. He is subject to colds and has visited his physician 5 or 6 times yearly for this condition. X-ray examinations and fluroscopic studies have always been supporting evidence of "bronchial trouble." The patient visited a chest clinic about May 1st of this year at which time a temperature of 99.4, rapid pulse, and flushed face were noted. He gave a history of cough, expectoration, intermittent attacks of shortness of breath and tightness in chest over a long period of time.

The physical examination at that time revealed evidence of cavity formation, left apex, and large bubbling rales over entire left chest. His family physician was informed that a diagnosis of far advanced tuberculosis was warranted, and sanatorium treatment advised. At this time, the patient was expectorating about four cupfuls per day.

Physical Examination

The patient is a fairly well-nourished young white man, who is mentally alert and does not appear acutely ill. His admission examination at the sanatorium revealed moderate clubbing of fingers; tonsils small; pharynx congested; teeth in good condition; vocal cords thick and red; voice husky. Respiratory rate 22 per minute, pulse 96, B. P. 90/70. See Plate I, Page 18.

Chest—Left

Some drooping of left shoulder and flattening of this side, anteriorly. Ribs, clavicles and other bony prominences conspicuous. Diminished expansion. Apex beat 1½ cm. outside mid-clavicular line but in fifth interspace.

Palpation

Tactile fremitus increased over some areas and decreased over others. No cariac thrill.

Percussion

Slight dullness over entire left chest, front and back. Left border of heart outside mid-clavicular line.

Auscultation

Amphoric respiration over apex, front and back, and over small area opposite the eighth thoracic spine in back. Areas of bronchial breathing over other parts of this side, front and back. Breath sounds diminished in some areas. Voice sounds accompanying above as expected. Large moist rales over entire left chest.

Chest—Right

Scoliosis toward right. Hyper-resonance and compensatory breathing on this side. Few large rales at base, front and back.

X-ray Findings, 5/13/36

Interpretations by Dr. L. R. Broome.

R. L.—Massive mottled density in root region extending into base. Slight haziness along trunks in other parts. Costophrenic angle obliterated.

L. L.—There is generalized thickening of the pleura and the chest on this side is flattened by inward drawing of ribs. Heart shadow appears shifted toward left and apparently it is hiding some of the lung structures. Lung shows some stringy density in root region extending into all parts chiefly the base. Along periphery throughout there are very thin-walled areas containing little or no lung markings. See Plate II, Page 18.

Summary—Findings on right suggest bronchiectasis. Those on left suggest a cystic lung, together with some inflammatory process which is present chiefly in the base. Findings on this side may be due to tuberculosis, with the annular shadows along periphery representing pockets of pneumothorax.

Lipiodol Injected for Bronchogram
5/19/36

The pharynx and larynx were anaesthitized with a 2 per cent solution of butyn. A soft rubber catheter was introduced into the trachea and forced down to the bifurcation. The patient was then placed on the fluroscope table, turned on his left side, and 20 cc's of lipiodol (40 per cent Iodine in poppyseed oil) given slowly through the catheter. Fluroscopic examination immediately afterwards revealed the left bronchial tree well out-

lined except for areas or "puddles" where the oil had collected. These were interpreted as dilatations or cavities. The bronchi of the apex were partially filled. They seemed to circumscribe a cavity which was well outlined. No oil entered this cavity. The cavities seemed to contract and expand with each respiratory excursion. The heart was located well over on the left side. An x-ray was made following the lipiodol injection. Because of cyanosis and dyspnea resulting from the diagnostic procedure, the patient was placed in a postural drainage position permitting much of the lipiodol to drain from his lung. He was further requested to lie on the bed with his head near the floor for 5 to 10 minute periods twice daily. Sixteen days later very little lipiodol remained. I have required my patients who were given lipiodol injections to assume this postural drainage position, and in all cases very little, if any, evidence of lipiodol was found after a few days. I wish to recommend this procedure for your consideration.

X-ray following Lipiodol, 5/19/36

Left chest—Films show material in the left lung. Dilated bronchi shown in middle part, while in the lower there are numerous areas indicating cavities, chiefly small ones. See Plate III, Page 18.

X-ray sixteen days after injection of Lipiodol, 6/4/36

When compared with 5/19/36, there is practically no lipiodol present now. See Plate IV, Page 18.

X-ray following Lipiodol injection into base of right lung, 6/5/36

Terminal bronchi in extreme base show definite dilatation, cylindroid and fusiform character. No saccules or cysts made out. See Plate V, Page 18.

Barium Meal

In order to rule out any possible diaphragmatic hernia, two glasses of barium were given orally. No filling defects nor abnormalities of the stomach and esophagus were noted.

Laboratory Findings

Sputum examinations: Three direct and three concentrated examinations were negative for tubercle bacilli.

Leukocyte count: On admission—12,600, with 78 per cent P. M. N. and 16 small lymphs, 5 trans., 1 eosinophil.

Leukocyte count: 6/24/36 — 23,600, with 88 per cent P. M. N. and 7 small lymphs, 4 trans., 1 eosinophil.

The last count was made merely as a comparison with the original. No symptons were found to account for the leukocytosis.

Wassermann and Kahn, negative. Urine examination, negative.

Sedimentation Test, See Charts VI and VII, Page 18.

An interpretation of these charts is difficult in that the second one (chart 2) portrayed an improvement in the patient even in the face of a high leukocytosis.

Tuberculin Skin Test: First dose P. P. D. negative at end of 24 and 48 hour periods.

Course in Sanatorium

Patient has gained in weight and strength. Sputum decreased from four cupfuls to ½ cupful per day. Generally improved.

Much has been said about the etiological factors, histological and pathological findings, the therapeutic problems and the need of a satisfactory classification of this pulmonary anomalous condition.

At the time of discharge the x-ray reading was as follows. See Plate VIII, Page 18.

X-ray Findings, 8/28/36

R. L.—Some thickening of trunk markings and haziness, especially those in lower half of lung, and there also appears to be a small amount of lipiodol still present. Costo-phrenic pleurisy.

L. L.—Has somewhat similar appearance as previous films, with the mediastinum and heart apparently pulled markedly to left.

Etiology

Many theories have been suggested for

the possible etiology of this congenital pulmonary defect. Since there are so many different opinions on this topic a discussion of them seems timely.

First, one might consider what is meant by cystic disease of the lungs. At present, most authors describe this anomaly as a localized distention of the bronchial tree, the walls of which may contain histological characteristics of the pulmonary unit. Others claim the defects may be congenital or acquired, and may arise from the bronchial epithelium or the interstitial tissues. Pearson[4] claims that those cysts which do not contain epithelium, cartilage, mucous glands, muscle and elastic tissue probably arise from faulty lymph anlage and therefore are lymphangiectatic spaces.

Lilienthal's[5] patient, age 54, had a cyst, the wall of which was devoid of epithelium but contained hyaline connective tissue. He thought he was dealing with an interstitial type. However, most cysts are lined with epithelium and, if elastic and thin-walled, may reach an enormous size such as the ballon cyst of childhood.

The congenital origin may be proved by finding pulmonary cysts in the stillborn and young infants. The greatest number may be congenital in origin but, as Pearson[4] claims, some develop after birth in defective areas of the lung or after some obstruction causes a distention of a portion of the bronchial tree distal to the obstruction. It is claimed by Muller and reported by Pearson that from pathologic studies a distention among congenital bronchiectasis, lung cysts, "honeycomb lung," small cystic degeneration of the lung, congenital vascular changes, fetal atelectasis, bronchial and lung adenoma and congenial lymphangiectasis is often impossible. A hydropic collection resulting in bronchial dilatation and eventually cystic formation may occur. Parmelee[6] and Apfelbach thought maldevelopment of the alvevlor tissue in that area was improperly supported.

Another theory of notable mention is that a cavity may form or result from a partially obstructed bronchus permitting inflation but preventing deflation. This theory would account for those single cysts communicating with the bronchi but not for those without bronchial communication, nor would it likely account for the multiple type. Some theories go so far as to suggest an inflammatory process during fetal life resulting in atelectatic bronchiectasis with improper inflation of the lung tissue at birth, and eventually terminating in destruction and cystic formation.

Diagnostic Difficulties

In diagnosing a cystic disease of the lung many difficulties are encountered. However, a history of dyspnea, varying degrees of cyanosis, wheezing or cough at birth or during early childhood in the absence of an acute infectious process or a history of an aspirated foreign body should make one suspect a "tension" cyst. The history pictured is often one of spontaneous pneumothorax.

Chart of Chief Complaints

COMPLAINT	CASES	PERCENTAGE
Dyspnea	18	26.8
Dyspnea & Cyanosis	10	14.9
Cough & Expectoration	12	17.9
Hemorrhage	8	11.9
Pain	4	5.9

The most frequent chief complaint occurring in the 67 cases reviewed was dyspnea. The symptom may make its appearance at birth or late in life, suddenly or insidiously, constantly or intermittently. As shown in the above chart 18 cases or 26.8 per cent had shortness of breath as a chief complaint, while 10 cases or 14.9 per cent claimed dyspnea and cyanosis as the first difficulty. More than 68 per cent stated that they experienced air hunger at some time during their illness.

The dyspnea may be attributed to one or more causes. First, the solitary cyst may reach such an enormous size or, if of the multiple variety, may occupy such a large pulmonary area as to cause a paucity of lung tissue. Second, the cyst may encroach upon adjacent structures

leading to atelectasis, kinking of the large bronchi, angulation of the trachea, displacement of the mediastinal contents and elevation of the intravenous pressure. Rupturing of a cyst or cysts, if located peripherally, often account for spontaneous pneumothorax. In fact, the most frequently mistaken diagnosis for cystic disease is spontaneous pneumothorax. Incidentally, the author has seen many cases of the last named condition without clinical symptoms.

Inflammatory conditions such as pneumonitis and influenza associated with cystic disease will lead to dyspnea in a very large percentage of cases.

Twelve cases or 17.9 per cent had cough and expectoration as the outstanding symptoms. As a rule the cough was productive and frequently offensive, especially in the long-standing infected multiple type. Seldom was there gross expectoration in the balloon cyst of childhood.

Hemorrhage may occur in the single cyst type of adults but rarely in the balloon cyst. Eight cases or 11.9 per cent of the series had pulmonary hemorrhage as the chief complaint, and 13 patients reported "blood spitting" at some time during their illness. The hemorrhages were usually moderate in quanity.

Pain in the chest occurred less frequently than any other symptom, only 4 cases or 5.9 per cent gave pain as the chief complaint.

A very large percentage, 16.9, gave no history suggesting lung cysts and the finding of the condition was purely accidental.

In other cases, signs of spontaneous pneumothorax, cavity formation, emphysema, consolidation, and fluid formation are found in the same patient.

With the absence of a family history of tuberculosis, a negative sputum, negative intradermal tuberculin test, the absence of foreign body aspiration, and, in place of the above, a history of intermittent dyspnea, cyanosis, and cough over a period of time, one should suspect "lung cyst."

The following chart reveals the part or parts of the lungs most frequently involved:

Chart of Involvement

LUNG	CASES	PERCENTAGE
Right	29	43.2
Left	23	34.3
Bilateral	15	23.0

The entire right lung was involved in 11.9 per cent of the cases but the left lung was not affected.

Involvement Confined to Specific Lobes

LOBES—RIGHT LUNG	CASES	PERCENTAGE
Upper	9	13.4
Lower	7	10.5
Middle	3	4.4
LOBES—LEFT LUNG	**CASES**	**PERCENTAGE**
Upper	8	12.0
Lower	9	16.4

The entire left lung was involved in 12 per cent of the cases at which time the right lung was not affected.

X-ray Findings and Interpretations

Freedman[7] points out that the roentgenological appearance is more characteristic than the symptomatology but, even so, many mistaken diagnoses of this condition are made. These cases are frequently called pulmonary tuberculosis, spontaneous pneumothorax, lung abscess, pneumonia, bronchiectasis, emphysema, and other sundry conditions. None of Koontz's 108 cases were diagnosed during life. Approximately 10,000 patients have been admitted to this institution (Catawba Sanatorium) during the past 27 years; yet the case reported herewith is the first congenital cyst recognized. However, we probably have failed at times in the proper diagnosis.

Let us first consider the solitary or balloon cyst of childhood. The clinical history will often help in the differentiation from the aspiration of a foreign body, gun shot or stab wound, or a spontaneous pneumothorax. Nevertheless, even with a history and all other available data per-

taining to the case, a definite diagnosis is impossible.

The cyst may occupy the entire hemithorax and even herniate through the anterior superior mediastinum to the opposite side. The wall of the cyst may be seen overlying the pericardium. Occasionally large dense shadows may be seen which subsequently disappear leaving large air spaces.

The following differentiations may be noted between balloon and solitary cysts and spontaneous pneumothorax:

Balloon and Solitary Cysts	*Spontaneous Pneumothorax*
1. Air cavity within lung.	1. Air in pleural cavity.
2. Wall of cyst not always detectable.	2. Pleura (visceral) generally seen separated from chest wall by air.
3. Normal lung workings may be found between cyst wall and periphery of lung.	3. Normal lung workings absent between visceral and parietal pleura.
4. Medastinal structures (especially in balloon type) may be displaced to opposite side.	4. Medastinal structures frequently displaced to opposite side.
5. Solitary type of adults may contain fluid but balloon type rarely contains much fluid.	5. Probably more than 95 per cent of these cases have fluid during their course.
6. Cavity of cyst remains relatively stationary.	6. Change in border of lung as intrapleural pressure changes.
7. Lung tissue generally found at apex and pericardiophrenic and costo-phrenic angles.	7. Lung tissues not found at apices and angles unless adhesions present.
8. Thin balloon cystic wall may be difficult to see.	8. Border of lung can generally be seen.

There are apparently many more differentiating points.

The following differentiation may be noted between multiple cysts and spontaneous pneumothorax:

Multiple Cysts	*Spontaneous Pneumothorax*
1. Walls of cysts frequently thin and vary in size, shape and location.	1. More or less regular pleural outline.
2. Cyst walls give an impression of a "pushing out" effect.	2. Lung conveys impression of intrapleural pressure.
3. If cysts are long-standing, mediastinal structures located on affected side as result of atelectasis and compensation of contralateral lung.	3. Structures generally forced to opposite side unless held by dense adhesions.

It is often difficult or impossible, clinically, to distinguish between cyst of the lung and tuberculosis with cavity formation. In the latter the inflammatory condition surrounding the cavities and the thickness of the walls of the annular shadows contrast with the thin-walled noninflammatory condition of the cystic formation.

Pleural rings may be ruled out by introducing a small quantity of air into the pleural space. If a cavity is present its shape and position generally change while a pleural ring remains unaltered. Of course, if the sputum is repeatedly positive the diagnosis may be simplified; however, a congenital cyst may be found in a tuberculous lung.

If bronchiectasis is suspected the same principles are applied. If the bronchiectasis is long-standing, with thick walls and a variable quantity of sputum, it may be impossible to differentiate it from an infected cyst of long-standing. Lipiodol injected into the diseased areas may give valuable information. I shall not discuss the differential points of cystic disease and lung abscess, ademona of the lung, etc.

Bronchoscopy may aid in the diagnosis but serious accidents have occurred which, to say the least, make this procedure questionable.

Physical Examination

In those patients with the balloon or tension type of cyst the physical signs are those of "spontaneous pneumothorax." Even in the solitary type of adults or in the large cavities of the multiple type, the physical examination will often reveal the same signs as spontaneous pneumothorax. However, large moist rales are frequently heard in the areas of small infected cavities. Often in the multiple type the physical examination may reveal very little, especially if the cavities are cen-

trally located.

Thirty-four cases were males and thirty-three cases females. There were also the following race difference:

Race			
White		*Colored*	
CASES	PERCENTAGE	CASES	PERCENTAGE
63	94	4	6

18 cases or 27 per cent were diagnosed during the first year of life. The youngest was a stillborn of 6 months and the oldest 67 years. The average age was 19 years.

Clubbing of fingers was not a frequent finding.

Thirty-eight cases, or 56 per cent, of the series were of the multiple type and 29 cases, or 43 per cent, were of the solitary type.

Treatment

In balloon cysts the treatment depends on the age of the child and the severity of the respiratory embarrassment. If the child is very young, aspiration of air is generally the preferred treatment. Occasionally foreign substance such as poppy-seed oil and formaldehyde have been used to obliterate the cavity. If the child is old enough, surgery may be considered.

The multiple type may not require treatment if no respiratory symptoms are present. As in the case of balloon cysts, if the cysts are large and rupture into the pleural cavity, air or fluid may have to be removed. The one-way valve mechanism used by Croswell and King has been used successfully in a few cases.

Bronchoscopy is seldom warranted. In the infected type, postural drainage seems of value.

Freedman claims that the infected cysts, if of the multiple type, should be opened widely and drained, providing symptoms are present. These patients should be cautious about contacts with acute infectious diseases. Infected tonsils and decayed teeth should be removed. Draining sinuses should be given attention.

I wish to thank the following physicians for their suggestions:

DR. J. B. NICHOLLS
DR. L. R. BROOME
DR. L. W. GARST
DR. A. T. COLE

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(Please turn page for Plates and Charts)

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Plate I

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Plate III

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Plate V

Plate VIII

BLOOD SEDIMENTATION CHART
Horizontal Line
Diagonal Line
Diagonal Curve
Vertical Curve
Normal or Quiescent
Quiescent to Slightly Active
Slightly to Moderately Active
Moderately to Markedly Active
TIME IN MINUTES
INDEX

BLOOD SEDIMENTATION CHART
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TIME IN MINUTES
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Clinical Aspects of Pneumonoconiosis *

BY
ROSS K. CHILDERHOSE, M.D.
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THE irritating effects of inhaled dust have been a widely discussed subject in recent years. The popular interest, not only in professional circles but also among the laity, has forced or is forcing new compensation laws in most of our states. Because of this increasing importance it is necessary for us as physicians to have a thorough knowledge of the subject so as to be able to treat appropriately those unfortunate patients who have a well developed pneumonoconiosis.

This disease has been somewhat confusing because of the numerous terms applied to dust destruction of the lung. The broad term covering all of them is Pneumonoconiosis. Such appelations as Anthracosis, Silicosis, and Asbestosis refer specifically to the type of offending dust. It happens in some industries, however, that workmen are subjected to a mixture of dusts and then the term "Pneumonoconiosis" is preferable. The normal protection offered by the ciliated epithelium of the trachea and bronchi is sufficient to prevent or delay the development of the disease where the concentration of dust is slight or even moderate. However, should the cilia be damaged by repeated respiratory infections or by irritating gases, then pneumonoconiosis will appear at an earlier period than expected and will be more extensive than under similar conditions.

Dusts may be classified under two groups depending upon their origin, i.e. (1) Organic, (2) Inorganic. Organic dusts do not cause pulmonary destruction, and only those inorganic dusts that contain silica or silicates can be definitely stated to be offending agents.

So widespread is the use of silica and silicates in industry that one should be familiar with their occurrence. The most important of the silicates is asbestos and in recent years the asbestos industry has come to realize that it is confronted with its own particular dust hazard, "Asbestosis." The danger is found particularly in the weaving process where the continued inhalation of the dust causes the development of an interstitial fibrosis in the mid-zone of the lungs. The disease is somewhat milder and does not develop as rapidly as that produced by silica. Silica is of prime importance in the mining of gold, silver, lead, iron, and anthracite coal. These minerals are found in rock containing silica and the rock dust constitutes the hazard. Similarly, quarry workers and those employed in dressing granite and sandstone are equally subject to this hazard. This would include sand blasters and foundry workers where castings are made. For many years it was thought that metal grinders and tool sharpeners developed a pneumonoconiosis because of the metal dust. We now know that it is not the metal dust that does the damage but rather it is the silica dust from the sandstones. Where carborundum wheels are used the hazard very materially decreases or entirely disappears. The manufacture of scouring and abrasive soaps requires silica in a very finely divided state and unless proper prophylactic measures are taken the hazard is serious. Similarly, the manufacture, grinding and polishing of glass may carry with it the serious possibility of pneumonoconiosis.

The factors controlling the development of the disease are few but important. Primarily, the worker must be exposed to a dust that contains more than ten million particles per cubic foot, and of which the silica content is thirty-five per cent or more. The conditions in different industries vary, but where the dust contains a high percentage of silica, such as in sand blasting and gold mining, the hazard is extremely high. This factor combined with the length of time to which

*Read at the meeting of the American College of Chest Physicians, Atlantic City, N. J., June 7, 1937.

the worker is exposed constitute the main elements. In the abrasive soap factories the hazard is greater because of the presence of an alkali soap powder that seems to facilitate the chemical change of the silica after it has been inhaled and thus to enhance its destructive effect. In the hard coal industry the average miner will not develop symptoms until after twenty years or more of work. However, if the anthracite miner has used the dry drill then the rock dust (thirty per cent silica) which he inhales will cause a third stage pneumonoconiosis in four or five years. In this connection let me state that the coal dust in itself does not cause any pulmonary destruction but merely pigments the lung. Any fibrosis in a coal miner is due to the contaminating rock dust.

In order to have a clear understanding of the signs, symptoms and prognosis of this disease it is necessary to explain briefly the mechanism whereby the lung is disabled and how eventually it may be completely destroyed. Essentially the disease is a disturbance of the lymphatic drainage of the lung due to the formation of scar tissue. The normal lymphatic drainage is inward to the hilum. A small area beneath the visceral pleura is drained into the sub-pleural cistern and from thence along the lobar septa to the hilum. When the dust particle reaches the alveolus it is picked up by alveolar phagocytes called "dust cells" and carried to the nearest lymphatic channel and there the process of lung destruction begins. The tiny dust particle, acted upon by the alkaline body fluids, forms a colloidal silica hydroxide which is a strong protoplasmic poison and by its action causes death of the dust cell and necrosis of the immediate surrounding tissues. A small scar is formed and the lymphatic channel is blocked. Should the dusting be relatively light the action begins principally in the hilus lymph nodes and later in the numerous small lymphoid deposits in the lung. If the dusting is intense the lymph derangement is so acute as to halt most of the dust in the mid-zone of the lung. As the dusting continues these minute areas of destruction (which are replaced by scar tissue) enlarge and we have nodular areas of fibrosis scattered largely in the mid-zone equally in both lungs. In the late stages these nodules coalesce to form massive areas of fibrosis that may ultimately replace ninety per cent of the lung. There are two other points of importance in this picture of pathology. The first is the compensatory emphysema that surrounds each area of fibrosis and which thus masks for the most part any physical signs of these fibrous consolidations in the chest. The other is the strain put upon the right heart by the obliteration in part of the pulmonary vascular bed. This extra work on the right ventricle usually terminates the picture in the last stages of pneumonoconiosis.

There are no characteristic stethescopic findings. In fact the physical examination of the chest is most disappointing. Even where there are large masses of fibrosis present the signs are practically non-existent. This is due to the compensatory emphysema that masks and prevents any exact estimation of the stage of destruction. No reliance must be placed on the physical findings. Clinical symptoms do not occur until the disease is at least moderately advanced. In the later stages we find dyspnea on moderate exertion perhaps combined with a slight unproductive cough. Weakness is prominent in the terminal stages and the dyspnea has now become orthopnea. With such few physical signs and symptoms, how then may the diagnosis be made?

The diagnosis rests upon the combination of a very carefully taken history and the roentgenogram. The history should include a description of the conditions under which the man works, the type of dust to which he is exposed, and an exact statement as to the length of time he has been in this dusty occupation. The interpretation of the roentgenogram requires experience in such work. The pictures readily show the fine nodular areas of the earlier stages in the lung to the large masses in advanced cases. The knowledge derived from the history will enable the roentgenologist to make the diagnosis quite easily.

A discussion of pneumonoconiosis would not be complete without a word as to its relationship with tuberculosis. It is generally accepted that the presence of silicosis predisposes the worker to tuberculous infection. This is particularly true under those conditions where the dust is practically pure silica. Amongst the hard coal miners of Pennsylvania this complication is not unduly prominent and the type of tuberculosis developed is one of extreme chronicity. A differential diagnosis is sometimes quite difficult and frequently depends upon a careful and prolonged study of the sputum.

One of the unfortunate features of silicosis is the tendency for the disease to advance even after the man has been removed from his dusty occupation. I have seen the fibrosis continue to increase in many miners long after they had given up mining. The proper treatment, therefore, for this condition is prevention. The application of suction devices to remove dust from its source has proven to be the most effective. Small face masks containing sponges are worse than useless because of their lack of protection and the false sense of security that they engender. After the disease is advanced the worker should, of course, be removed from his occupation and given a thorough bed rest so as to relieve the right heart and increase the cardiac reserve. The course of the disease should be followed at all times by frequent roentgenograms.

Echoes and Reverberations

(A report on the National Tuberculosis Conferences with an analysis of the Early Diagnosis Problem)*

BY
CHARLES I. SILK, M.D.
Perth Amboy, N. J.

THRUOUT the entire proceeding of the annual meetings of the American Sanatorium Association and the National Tuberculosis Association held in Milwaukee, as well as at the meetings of the Federation of American Sanatoria (now the American College of Chest Physicians) and the American Academy of Tuberculosis Physicians, including the Symposium on Tuberculosis of the A. M. A. held in Atlantic City, the one dominant note which kept constantly recurring like a fugue in a first rate musical composition of Bach or Handel was that of Early Diagnosis. Not that the facts brought out were new, but they were sufficiently emphasized to make them stand out in bold relief like a sore thumb. To wit: 57 per cent of all admissions to the Tuberculosis Sanatoria in the United States are in the far advanced stages of the disease, 30 per cent are moderately advanced and only 13 per cent are in the minimal class. The family physician is responsible for 65 per cent of all admissions. Everybody agreed that something must be done about it.

Early Case Eludes

When we consider that in spite of the advances made in diagnostic procedure in the past quarter of a century, the great effort put forth and the large sums of money expended in this direction for this very purpose, and still find that only 13 per cent of all sanatorium admissions are in the minimal stages, and consider the further fact that in this minimal group are to be found most of the wrongly diagnosed cases as well as the cases that need no hospitalization because their lesions represent benign or healed pathological processes, we have a picture not very pleasant to behold. Everyone realizes that if further substantial results are to be achieved in the reduction of tuberculosis mortality, or in the incidence of its mor-

*Read at the July meeting of the Executive Board of the New Jersey Tuberculosis League, 1937.

bidity, and particularly in the control of the disease, we must find our cases much earlier than we do at the present. This, of course, is easier said than done.

Remedial Measures

The remedies suggested were many, most of which were very good while others perhaps not so good: Raising the standards of medical education, stressing the value of the x-ray picture and the tuberculin test in the diagnosis of pulmonary pathology, particularly that of tuberculosis, arousing the interest of the family physician in tuberculosis, making him tuberculosis minded, reducing the cost of the x-ray film, educating the public to be tuberculosis minded, periodic health examinations, examinations of contacts, mass examination of school children, higher age groups, lower age groups, preschool children and infants, adult groups, food handlers, industrial groups, school teachers, college students, institutional population, colored population, substitution of the fluoroscope or paper film for the expensive celluloid film, etc., ad infinitum etad nauseam. These are some of the more important suggestions advanced for the finding of early or minimal cases of pulmonary tuberculosis.

Diagnostic Difficulties

Before evaluating these various suggested measures for the discovery of minimal cases of pulmonary tuberculosis it may be well to consider some of the basic data available. First of all we must bear clearly in mind that there is a marked difference between early diagnosis and the diagnosis of early or minimal pulmonary tuberculosis, that the term minimal as defined in the Diagnostic Standards of the N. T. A. serves as a quantitative measure only of a given lesion and does not indicate whether or not it is active or progressive or both, that the factor of personal equation enters largely into the making of such a diagnosis, that as a consequence our statistical data must suffer a considerable error in this respect, that many active minimal lesions do not progress, do not caseate nor cavitate, do not present open lesions with tubercle bacilli in the sputum. On the other hand, some of the minimal cases progress rapidly with caseation and cavitation, with tubercle bacilli in the sputum, sometimes even within a few weeks, and many apparently healthy persons may have advanced disease without symptoms or even physical signs. It is these considerations that make the diagnosis of early cases so complex and difficult.

Family Doctor

Concerning higher standards of medical education and the education of the family physician, we wish to make the following comments. There are approximately 70,000 deaths annually from all forms of tuberculosis in the United States. Allowing ten possible active cases for every death, we have about 700,000 active cases at any one time. There are also 170,000 practicing physicians in all branches of medicine in the United States. This gives a ratio of 4.11 possible cases that the average physician may see in any given year if all cases presented themselves for diagnosis and care. If we deduct from this 25 per cent of all cases who seek the clinic for free advice and another 25 per cent who never seek any physician's advice because of lack of symptoms, ignorance or poverty or any combination of these, there remains only one-half of all possible cases, or two patients per physician. The point we wish to make is to the effect that no amount of education or training can keep a physician expert in the diagnosis of any disease of which he sees only two cases a year at the utmost. The family physician however is in a key position for the solution of the early diagnosis problem and his services can be enlisted in the fight against tuberculosis by: 1st, continuously reminding him of the importance of tuberculosis as a public health menace because of its communicability, insidiousness, chronicity and long invalidation of its victims with all its social and economic implications as well as of the high mortality, thus making the physician tuberculosis minded; 2nd, having made him tuberculosis

minded it will be necessary to place at his disposal x-ray facilities at a reasonable rate or free, as the case may require, laboratories for sputum examinations including a nursing follow-up service—in other words a chest diagnostic and consultation clinic where he can refer his patients for necessary information and diagnostic service without losing contact with his client. Only then can we expect his whole-hearted cooperation.

Diagnostic Service

Irrespective of the skill and knowledge a physician may possess, it is unreasonable to expect him to do full justice by his client in whom a minimal type tuberculosis is suspected when we consider the time and effort required in taking a careful history, making a complete physical examination, tuberculin testing, x-ray examination and interpretation, the repeated collection of sputum specimens, the clinical observation by means of pulse and temperature chart for a period of ten to fourteen days, and the compensation he can command for such services unless a diagnostic service such as outlined above is made readily available to him.

Public Education

The public must be educated to the need of periodic examinations including an x-ray film or a fluoroscopic examination of the chest. The public must also be made to realize that an apparently healthy individual may have extensive pulmonary tuberculosis without symptoms and that only by means of tuberculin test and x-ray examination can this be revealed.

Open Adult Case

Sufficient data has been presented to make it crystal clear that we must shift our emphasis from the school child to the adult where the greatest number of open cases of tuberculosis are to be found. Instead of bending every effort to finding the infected child, it is far more logical and should prove by far more profitable to seek out the adult who is the real source of the infection. Dr. H. R. Edwards of New York City reports 20 per cent manifest lesions in adults against 1.4 per cent in children, Dr. William P. Brown of Albany, N. Y., 4 per cent in adults, ½ per cent in children. Dr. George W. Webber of Ulster County, N. Y. found 2½ times as many cases among parents as among children. This should be proof enough to confirm the above contention. Dr. J. A. Meyers of Minneapolis says, "We have known for a long time that there is far more tuberculosis in persons in the twenties and thirties than in the teen age period." How to reach the adult group is the major problem of the day.

Productive Groups

Beginning with the higher teen ages which can and are being reached in the schools and then followed up in the colleges and various institutions of secondary education, teachers in all institutions of learning as well as other school personnel, all public employees whether municipal, county, state or federal, all hospital personnel including doctors and nurses, all patients admitted to general or special hospitals for whatever reason, all inmates of institutions (penal, correctional, insane, epiliptic, etc.), relief families, colored groups, contacts, food handlers, domestics, etc.—all these constitute fruitful fields for tuberculin testing and x-ray examination. If such procedure were carried out persistently over a sufficiently long period we would not only reach a large number of adolescents and adults but it would result in a health consciousness on the part of the public, demanding these services as a routine part of its daily life.

Recommended Program

It should be perfectly feasible to formulate a program which is practical in its application in almost any community based on the foregoing considerations, somewhat as follows:—

1. An educational campaign with a view of making the family physician tuberculosis minded by continuously reminding him of the seriousness and importance of tuberculosis as a public health

and social menace; that a physical examination, no matter how skillfully performed, cannot be relied upon for the discovery of early pulmonary tuberculosis; that tuberculin testing and x-ray examination of reactors is highly efficient.

2. The establishment of diagnostic and consultation chest clinics where the physician may feel free to refer his suspect without fear of losing contact with his patient.—This service to be furnished at a nominal cost or free, as the case may require.

3. Education of the general public, stressing the fact that an apparently healthy person may have serious tuberculous lesions of the lung, necessitating periodic health examinations to reveal them; that this must include an x-ray film or a fluoroscopic examination of the chest unless the need for it is ruled out by a negative tuberculin reaction to I. mgm. of tuberculin.

4. A concerted effort at reaching the adolescents in schools and young adults in all institutions of secondary education, all teachers and school personnel, all public employees, all hospital personnel, all patients admitted to hospitals, all inmates of public and private institutions, food handlers, domestics, contacts, etc.

So much for early diagnosis which we consider the most important problem in tuberculosis control and the solution of which needs our most serious and careful consideration.

New Serum Test

The pathological section offered a new serum test, "The Weltmann Serum Coagulation Test in Tuberculosis," which promises to be of practical value as a non-specific prognostic indicator, claiming to be superior to the blood sedimentation test or the white blood count.

Surgical Techniques

The surgical section was mainly concerned with improved technique in thoracoplasty, particularly the new method of apicolysis which offers better results and a lower mortality. The social work section debated the controversial subject of the use of preventoria. Dr. C. A. Stewart believes that preventoria should be converted into hospitals for patients with serious disease in its communicable stages. The question of finding tuberculosis among apparently healthy adults was also discussed.

Rehabilitation

The administrative section had as an outstanding subject for its consideration, the rehabilitation of the tuberculous. Although very little is being accomplished along this sector of the tuberculosis war front, Dr. Beulah W. Burhoe tackled the problem for the first time in a more realistic way by recognizing it as chiefly a social and economic problem. As soon as we are ready to subsidize the tuberculous handicapped and provide him with working conditions under medical supervision, commensurate with his disability, we will make progress, but not sooner.

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VITALY J. ALEXANDROV, M.D.
Rutland, Massachusetts

TUBERCULOSIS is not a new disease. It is probably one of the oldest known, and is mentioned as a communicable malady as far back as 400 B. C. Hippocrates described cough, expectoration, chills, fever, night sweats, ischiorectal abscess, loss of weight, dyspnea, fatigue, and G-I symptoms.

In the 1680's, Morton recognized tuberculosis of the lungs, throat, and G-I tract. He advised examination of the lungs and throat when a patient complained of G-I symptoms.

In 1800, Bayle stated that tuberculosis was infectious. He said that caseous material thrown off spread the disease, as we know to be true. It was Bayle who gave the name tuberculosis to this disease.

Our present knowledge, progress of treatment, and prevention began in 1882, when Koch discovered the tubercle bacillus, and proved that this bacillus was the cause of tuberculosis. At that time, early diagnosis would not have had the importance that it has today. Little was known about treatment and it is doubtful whether or not anyone thought about prevention at that time.

Now we have everything to work with. There are laboratories, x-ray, plain and stereo; there is the tuberculin test and the stethoscope. Today we have a scientific and intelligent understanding of the symptoms of tuberculosis.

Our problem now is how best to apply our present knowledge to the advantage of those who are suffering, or might be suffering, from the disease.

In the case of a non-tuberculous patient, we usually think about him and his illness as the sole problem; but in a tuberculous case, it is impossible to think about the patient alone, since the family and community are also involved. Therefore, as soon as a diagnosis of tuberculosis is made, we should tell the patient about it and immediately arrange for a check-up of his family, at the same time taking steps to safeguard the health of the community.

We need to examine the family so that we can find the sources of infection and be cognizant of their present condition. When we find that one or more members of a family have tuberculosis, we take care of them as well as if the patient who came to us first. In doing these things, we safeguard the health of the community by isolating the open case of tuberculosis.

It is for these reasons that tuberculosis is a very serious and complicated problem. Only by a good understanding and a sympathetic approach will we be able to decrease materially the number of persons afflicted.

Besides detecting the disease in those who have it, especially in the open cases, and examining the patient's whole family and other contacts, we have an educational obligation. All open cases should be isolated, preferably in a sanatorium, and should be told of the danger of spreading the disease to their family, children, and community.

As a general rule, when we see a patient, his disease is so far advanced that an early diagnosis is impossible. However, if we examine his family and contacts promptly, we may feel confident that we have a better chance of securing an early diagnosis in these cases than if we waited for these same people to break down and come to us.

It is almost impossible to confuse a diagnosis in advanced cases, but in early and doubtful cases just the opposite is true. One great help in such cases is the family history of the patient, especially as regards to sources of infection. If a patient reveals that there has been some sort of lung trouble in his family, in father, mother, uncle, etc., it is advisable to go into detail and find out the duration of the illness, general condition, cough, expectoration, and weight. It is indeed surprising to find out how many negative family histories turn out to be positive

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ones.

Going into the past history of the patient, we should pay special attention to the duration of cough and expectoration and the so called frequent colds. If the cough lasts more than a month, we should consider the possibility of tuberculosis until there is proof to the contrary. Then there are also pleurisy and ischio-rectal abscesses which in a large percentage of cases are due to tuberculosis.

As to symptoms, the most important are fatigue, absence of appetite, irritability, weakness, night sweats, pain in the chest, insomnia, hoarseness, elevation of temperature with or without rapid pulse, loss of weight, and cough, dry or with expectoration.

We do not base our final diagnosis on either the history or the symptoms, but rather on an intelligent interpretation of both. In our interpretation of symptoms, both from the physical and laboratory findings, we should direct our attention to abnormal findings and attempt to find an explanation for them. If we can find, after careful investigation, no explanation for the patient's present condition, we should seriously consider the possibility of tuberculosis. Especially does this hold true when we find the patient's past history, or the history of the family, positive. In such a case, it becomes a moral responsibility to tell the patient that, while there were not enough findings to make a diagnosis of tuberculosis, his or his family's history, together with some symptoms present, make the presence of tuberculosis a strong possibility. We should further advise him to accept treatment for tuberculosis.

It would indeed be a great mistake to wait until we find all the symptoms and physical signs in our examinations. If we delay action until we have secured *all* the convincing data, the chances are that the disease will have already advanced to the second or third stage.

The family doctor, who, after all, sees the patient first, often overlooks a very important phase of the control of tuberculosis: that is, requesting the consultation of a tuberculosis specialist. This is strange, because the advice of the heart specialist, the eye, ear, nose, and throat man, the endocrinologist, etc., is invariably asked, when a case involving their specialty comes to us. Considering the fact that in the latter case the patient's health alone is concerned, while in the former, the health of the community and the patient's family, as well as the patient's health are concerned, I believe that the family physician should request a consultation in all doubtful cases, and sometimes in obvious cases where it would add to the patient's morale.

Now that we have completed our diagnosis, what are we going to do with those patients in whom a positive reaction has been noted? It is my belief that these patients should be sent to a sanatorium for a stay of three to six months, where they can receive all the benefits of modern methods of treatment, as well as an education which will teach them to take care of themselves and to prevent spreading the disease throughout the community.

If the physician advises home treatment in the beginning, he must take into consideration the surroundings, will power of the patient, his ability to follow orders, etc. In most instances, the physician will, I believe, find the obstacles too great and will advise sanatorium treatment.

If, in spite of these obstacles, home treatment is decided upon, all members of the family should submit to an examination which should include physical, tuberculin, x-ray, and sputum tests. These tests should be given at six-month intervals, and, if possible, the interval should be shortened to three months.

We all agree that if there are children in the home, a sanatorium should be insisted upon. The following survey gives ample proof of such a necessity: It shows that two out of every three children with tuberculous lesions of the adult type come from families where there has been an open case of tuberculosis; that the tuberculin test shows that 48.5 per cent are positive when there is contact; that 26.2 per cent are positive when there is no known source of contact. McPhedran shows that the disease amongst those who

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have a known contact ranges from 11.1 per cent in children up to five years of age, increasing to 70.6 per cent between the ages of fifteen and twenty years, as compared with 0 to 33.3 per cent where there is no known contact, during the same period of life.

This state of affairs, with "contacts" walking around the streets unsupervised, is fraught with extreme danger. If we stop to consider that there are about 90,000 beds in this country and that there are about 800,000 open cases of tuberculosis, most of whom are not under constant medical supervision, and who are without proper education regarding the danger of tuberculosis, we realize that the number of contacts these people make must be an enormous one.

To alleviate this condition, that is, to control Tuberculosis, certain steps are necessary. First, we must keep the disease in mind. It is our duty to take care of an open case, and to do so at once, not allowing him to establish contacts with the public until the sputum is negative. Second, we must examine the family of every case of tuberculosis and follow up these examinations at regular intervals for a number of years. Third, we must strive for early diagnosis. Not only is this last an important economic and life-saving measure, but early diagnosis means early isolation of those who are spreading the disease, early treatment, and early negative sputum.

The burden of all this rests mainly on one pair of shoulders, those of the family physician. It is not a job for the state legislature, or for the laity. Only by the unflagging interest of the family physician, the man who sees the patient first, can any sort of program be effectively put into operation. Thus the family physician will render one more great service to the community and to those who are suffering from tuberculosis.

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ORGANIZATION NEWS

Dr. Hayes Guest at Association of Medical Colleges Meeting

Dr. E. W. Hayes, Monrovia, California; President of the American College of Chest Physicians was a guest of the Association of American Medical Colleges at their forty-eighth annual meeting which was held at San Francisco, October 25-27. Dr. Hayes was invited to attend the meeting and to present a program for the more efficient teaching of chest diseases to Junior and Senior medical students. Last year, Dr. Hayes headed a Committee which conducted a survey of the teaching of chest diseases in the medical schools of this country and the results were presented at the annual meeting of the American College of Chest Physicians at Atlantic City in June of this year. The report was since published in the September issue of *Diseases of the Chest*. A copy of the report may be had by writing to the offices of the American College of Chest Physicians.

New Fellows

The Board of Regents announces the acceptance of the following physicians as Fellows in the American College of Chest Physicians since the publication of the last Pneumothorax Directory:

Dr. D. L. Alarcon, México City, D. F.
Dr. J. M. Appel, Cleveland, Ohio.
Dr. M. M. Baumgartner, Janesville, Wis.
Dr. T. C. Black, Norton, Kansas.
Dr. B. Bloom, Tucson, Arizona.
Dr. W. C. Breidenbach, Dayton, Ohio.
Dr. W. J. Bryan, Rockford, Illinois.
Dr. A. V. Cadden, Hopemont, W. Virginia.
Dr. M. Cañizares, Philippine Islands.
Dr. J. D. Davis, Los Angeles, California.
Dr. H. A. Gorman, Hamburg, Pennsylvania.
Dr. Israel Kaufman, Brooklyn, New York.
Dr. M. D. Miller, Columbus, Ohio.
Dr. L. D. Phillips, Marshallton, Delaware.
Dr. E. A. Schaper, San Leandro, California.
Dr. Jane Skillen, Olive View, California.

Dr. Bortree President-Elect, Colorado

Dr. Leo W. Bortree, Fellow American College of Chest Physicians, was unanimously elected as the President-elect of the Colorado State Medical Society at its Sixty-seventh Annual Session held at Colorado Springs. Dr. Bortree has been actively identified with tuberculosis activities at Colorado Springs since his graduation from the Harvard Medical School in 1910 and the Fellows of the American College of Chest Physicians extend their congratulations to him upon his elevation to the Presidency of the State Medical Society.

Society Notices.

Dr. William J. Bryan, Fellow American College of Chest Physicians, has been appointed as the Superintendent of the Municipal Tuberculosis Sanatorium at Rockford, Illinois. Dr. Bryan was formerly Superintendent of the Missouri State Tuberculosis Sanatorium, at Mt. Vernon, Missouri.

Dr. Harry C. Warren, Belmont, California; Fellow American College of Chest Physicians, addressed the Forty-third Annual Meeting of the Utah State Medical Association at Salt Lake City, Utah, September 2nd. The title of his paper was, "Surgery versus Conservatism in the Treatment of Pulmonary Tuberculosis."

Dr. Paul H. Ringer, Asheville, N. C.; Fellow American College of Chest Physician, read a paper before the Fifth District Medical Society, Atlanta, Georgia; on October 7th entitled, "Evolution of the Treatment of Tuberculosis."

Dr. Hyman I. Spector, St. Louis; Fellow American College of Chest Physicians and Tuberculosis Controller, City of St. Louis, addressed the meeting of the National Safety Congress at Kansas City, October 5th. The title of his paper was, "Industrial Tuberculosis—A Public Health Factor."

Dr. Jerome R. Head, Chicago; Fellow American College of Chest Physicians, presented a paper on "Thoracic Surgery" before the Fort Wayne Medical Society, Fort Wayne, Indiana, on September 14th.

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